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# Visceral leishmaniasis after cardiac surgery

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## Abstract

**An English child developed visceral leishmaniasis (kala-azar) after cardiac surgery. Neither he nor his mother had ever been out of the UK, and his disease was probably transmitted by blood transfusion. Kala-azar should be considered in patients with unexplained fever and hepatosplenomegaly, even if there is no history of foreign travel.**

(Arch Dis Child 1995; 72: 235-236)

Keywords: leishmaniasis, blood transfusion, cardiac surgery.

Kala-azar is caused by parasites of the *Leishmania donovani* complex and occurs widely in parts of Africa, the Mediterranean, Asia, and Central and South America.<sup>1</sup> The disease is spread to humans from the natural reservoir, usually a dog or rodent, by the bite of an infected sandfly, except in India where humans are the natural hosts; symptoms relate to multiplication of the parasite in the mononuclear phagocyte system. In non-endemic areas kala-azar is rare and largely confined to travellers who have visited an endemic region.<sup>2,3</sup> We report an English child who developed the disease without ever having left his native country.

## Case report

The child was born at 32 weeks' gestation and remained in hospital until the age of 2 years. At age 6 months he underwent surgery for absent pulmonary valve syndrome. Postoperatively he suffered from recurrent chest infections and anaemia that required frequent red cell transfusions. At age 18 months he developed fever that failed to respond to broad spectrum antibiotics. Physical examination revealed marked hepatosplenomegaly.

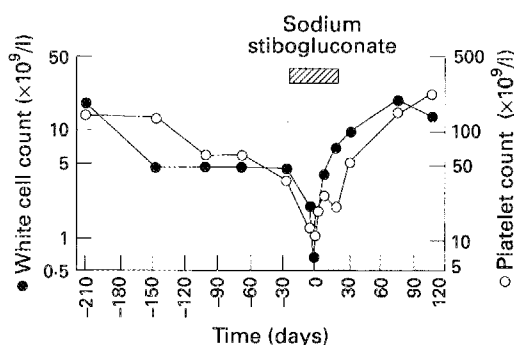
His haemoglobin concentration was 87 g/l, white cell count  $2.6 \times 10^9/l$  (10% neutrophils, 76% lymphocytes, 14% monocytes), and platelets  $26 \times 10^9/l$ . The direct antiglobulin test was weakly positive. Serum albumin concentration was 34 g/l (reference range 35-50 g/l), globulin 54 g/l (20-35 g/l), aspartate aminotransferase 56 IU/l (0-46 IU/l), alkaline phosphatase 910 IU/l (160-1150 IU/l), and bilirubin 15 mmol/l (2-17 mmol/l). Repeated cultures of blood and urine were sterile, viral serology was negative, and tests for tuberculosis and mycoplasma infection gave normal results. An echocardiogram revealed no valvular vegetations. A technetium labelled white cell scan showed increased uptake in

the spleen, consistent with hypersplenism. An abdominal computed tomography confirmed hepatosplenomegaly but was otherwise normal. His pancytopenia eventually prompted examination of a bone marrow aspirate. This was hypercellular and, surprisingly, contained numerous Leishman-Donovan bodies. The diagnosis of visceral leishmaniasis was confirmed by leishmania direct agglutination and indirect fluorescent antibody tests (titres 102400 and 30, respectively). He received 20 mg/kg sodium stibogluconate intravenously for 30 days, after which there was resolution of fever, a rapid recovery of white cell and platelet counts (figure), and clearance of the parasite from bone marrow.

The patient and mother had never been abroad and so it was felt the most likely source of the infection was transfused blood products. Before developing leishmaniasis the child had received 15 units of red cells and one unit of fresh frozen plasma. Fifteen of the 16 donors responded to a request for follow up: there was no history of foreign travel in six, and the leishmania direct agglutination proved negative in the other nine. Eight of the nine antibody negative donors were also tested by the intradermal leishmanin test and by buffy coat culture, all with negative results. Two other modes of spread were considered: transplacental and vector borne. The former was judged to be extremely unlikely and maternal serology proved to be negative. A bite from an imported vector, as described in airport malaria, seemed a remote possibility but the child had never been out of hospital and there have been no reports of leishmania being transmitted by this route in the UK.

## Discussion

In non-endemic parts of the world visceral leishmaniasis is seen mainly in patients who have (recently) visited an area of possible endemicity. We believe ours is the first



White cell and platelet counts before and after treatment.

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reported case of kala-azar in an English child who has never left his native country. An extensive search failed to identify the source of infection but it seems likely the parasite was transmitted by transfused blood products. Blood transfusion is widely recognised to pose a risk for the transmission of various infectious agents, most notably HIV, cytomegalovirus, and hepatitis viruses.<sup>4</sup> The full list of diseases transmissible by transfusion, however, is long and includes malaria, brucellosis, Lyme disease, trypanosomiasis, babesiosis, filariasis, and leishmaniasis.<sup>4</sup>

Haematogenous spread of leishmania was first described by Chung *et al* in two girls who received intramuscular injections of maternal blood for measles prophylaxis,<sup>5</sup> and has since been reported after blood transfusion in transplant recipients and newborn infants.<sup>6</sup> In endemic areas the large number of subclinical leishmania infections is sufficient to maintain interhuman transmission by this route in the absence of an animal reservoir.<sup>7</sup>

Because of scepticism about the diagnosis and inexperience with the disease, treatment of our patient was delayed for several weeks, during which time a number of unnecessary investigations were performed. The presence of fever, hepatosplenomegaly, pancytopenia, hyperglobulinaemia, and a positive direct antiglobulin test should always alert physicians to the possibility of leishmaniasis, particularly when there is a history of blood transfusion.

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